50 years on: the crush syndrome

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In this year of major earthquakes in Iran, Romania, the Philippines, and Peru—not forgetting a small one near Bishop's Castle in Shropshire—it is well to remember the events of 50 years ago when, during the Blitz and the Battle of Britain, many hundreds of civilians were buried beneath the debris of their own houses (fig 1). Much was learnt then and during the course of the war about what we called "crush injury" and later the crush syndrome[1,2] that is relevant in the rescue and treatment of earthquake casualties.[3] Fifty years ago bombing occurred in built up urban areas provided with a prepared and relatively efficient hospital service and affected few patients compared with the vast number in the often rural and neglected zones of tectonic plate collisions, where logistics loom large in rescue work.[4] Knochel, for instance, estimated that 80% of those affected die of head injuries and asphyxia, leaving only 20% of immediate survivors; half of these have uraemia.[5]

This paper presents a personal and very brief account of crush syndrome from 1940, seen in the light of later developments, enabling us to draw conclusions helpful today. Our first experience of this challenging type of injury came on 16 September 1940 at the Hammersmith Hospital in west London, then the British Postgraduate Medical School. John McMichael, reader in medicine, was in charge after Professor Francis Fraser's appointment as supremo of the emergency medical service. The team included Sheila Sherlock, Peter Sharpey-Schafer, Otto Edholm, Henry Bancroft, and Professor Gask: in the previous months of the "phoney war" they had undertaken a programme of work on shock and the physiological effects of blood loss and replacement using themselves as experimental subjects. This took on a more practical and serious aspect after the capitulation of France (10 May 1940) and the miracle of Dunkirk (26 May to 4 June 1940).

Britain was alone and Winston Churchill's call, "We shall fight on the beaches: we will never surrender," was echoed throughout the country. After Hitler's final demand for our surrender (19 July 1940) German attacks on airfields and docks preparatory to the invasion of England (Operation Sea Lion) were greatly stepped up. Although London had been spared so far, a retaliatory attack on Berlin ordered by Churchill (25 August) for some erring bombs on London a few days earlier brought the full force of Goering's Luftwaffe on London starting on 7 September. We at Hammersmith in Wormwood Scrubs were far away from the initial fiery holocaust in the docks and the east end, although we could see it in the night sky, but by Sunday 15 September a massive and more widespread attack was launched, in which, as was to be usual, far more enemy aircraft were brought down than those of ours. This and later raids resulted in saturation of the bed capacity of central hospitals and transfer of less urgent casualties to less central hospitals.

On 16 September we received several casualties at Hammersmith—the usual lacerations, burns, fractures, and shell shock—and also two patients with no apparent external injury.[1] They had been buried in the debris of their houses for a number of hours, and when dug out seemed to the front line casualty doctors to be unscathed and have a normal blood pressure. In the melée they received little attention for an hour or so, when they suddenly collapsed without sign of either external or internal bleeding but with gross limb swelling and skin wheals. Haemoconcentration was present, as was low blood pressure; this was restored to normal by serum and saline transfusions, but oliguria set in. Uraemia and death occurred on the 3rd day (case 3) and 6th day (case 4).[1] Two similar patients were admitted on 4 December. They were more carefully studied, were reported to the Medical Research Council shock committee, and the details published in March 1941 with 13 other cases from London.[1] Both died of uraemia, and more complete studies at necropsy confirmed not only muscle necrosis but major, specific changes in the kidneys.[6]

Many other casualty teams began to recognise similar cases, always after compression under debris for several hours. We assisted at necropsies in several such cases, occasionally having to shelter beneath the necropsy table: the results were notified in memoranda to the Medical Research Council's subcommittee on traumatic oedema, which first met in December 1940. Theories abounded and remedies too. The analogy, clinical and histological, with mismatched transfusion kidney was close. This had been characterised by Baker and Dodds in 1925 as due to haemolysis, with oliguria and acid urine leading to blood pigment obstruction of the renal tubules.[7]

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FIG 1—*Extricating buried patients during the Blitz (courtesy of Planet News Ltd)*

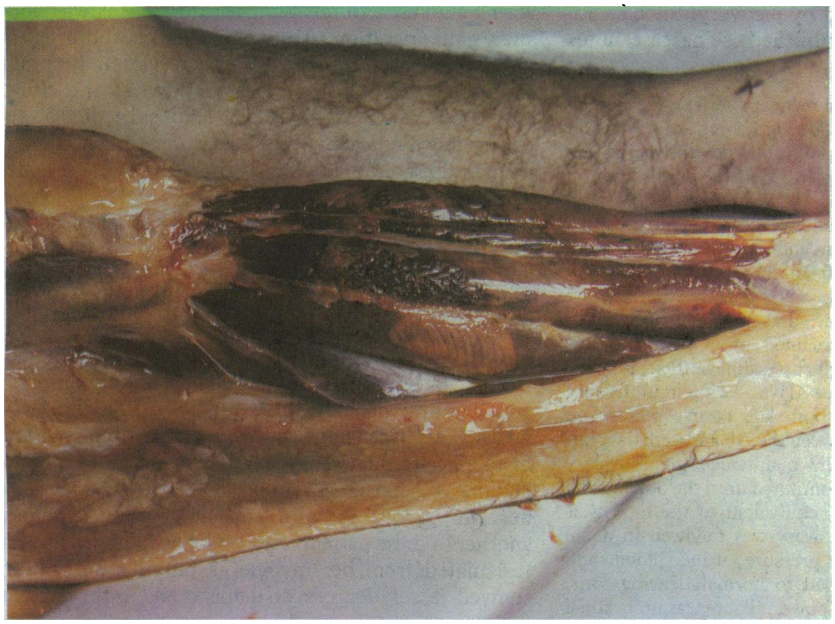

FIG 2—*Muscle of patient buried for six hours and surviving 7½ days showing oedema and necrotic lateral muscles of the leg (courtesy of Metal Box Company; reproduced from Bywaters and McMichael*[13])

Myoglobin was suggested as the important nephrotoxic agent,[1] and we soon showed this in urine as myoglobin, metmyoglobin, or finally acid haematin, and later even in serum.[8] We crystallised human myoglobin for the first time at Hammersmith, serendipitously having left a solution in the refrigerator overnight. The low renal threshold of myoglobin compared with haemoglobin made its detection in serum, except in the most severe cases, impossible at that time. Later technological advances and radioimmunoassay have made nanogram amounts detectable, even in normal controls, who have a regular circadian rhythm.

Back in 1941, when we had only the benzidine test and the spectroscope, we were able to clinch the Baker-Dodds haemoglobin hypothesis for myoglobin by producing muscle ischaemia in rabbits with a pressure bandage applied under anaesthesia.[9] These leg muscles contained no myoglobin, and we reproduced the "shock" phenomenon but not the renal consequences. The renal consequences were finally produced in rabbits by injecting human myoglobin solution—but only in acidotic animals, not in controls, whether compressed or normal.[10]

Thus the sequence of events began to be clarified. Air raid victims not dying from direct trauma, suffocation, blood loss, burns, or fat embolism, rescued from burial and compressed for more than two and a half hours (fig 2) suffered irreversible ischaemic muscle necrosis dependent to some extent on circumambient temperature as cooled tissue has a longer ischaemic survival time. When the victim was dug out, the returning circulation brought blood to the necrotic muscle. This absorbed sodium chloride, became swollen, and released myoglobin, lactic acid, creatine, creatine kinase, phosphate, and potassium into the circulation. Potassium disrupted cardiac rhythm, and calcium was later absorbed, leading to hypocalcaemia and sometimes local calcification. The released myoglobin filtered at the glomerulus into an acid and concentrated urine, precipitated in the distal convoluted tubules of the kidney, reduced reabsorption, and led to greatly increased intrarenal pressure (and of course many other mechanisms have been invoked). The kidneys that we examined were almost always overweight and oedematous (fig 3), immediately bulging out when the capsule was incised, except in patients who died early. The distal tubules ruptured into the interstitial tissue and into the renal venules.[6] These tubulovenous aneurysms are similar to the changes in acute hydronephrosis[11] and those in sulphapyridine and sometimes uric acid crystal blockage.

By July 1941 the temporary cessation of raids on London allowed us to look back at the records of the first world war. None of six British war surgery books published in 1918-9 nor any up to 1941 mention this syndrome, nor did the American medical history of the first world war.[12] It was, however, given a considerable mention, citing 126 cases, in the official *German Handbook on Medical Studies in the World War*.[13] Originally described by Von Colmers in the Messina earthquake of 1909, the syndrome was first reported in war injuries by Frankenthal in 1916 and followed by several other German publications, subsumed by Minami in 1923, who added three original case records. He suggested the possibility of myoglobin as a nephropathic agent. History, it is frequently said, teaches us that man does not learn from history. Although it may be true that the military machine begins war prepared for the previous one, the medical machine in 1939 was not as fully prepared for even a previous war as it might have been had it consulted its opponent's publications.

Treatment and prevention

By 1941, on the analogy of the mismatched transfusion kidney lesion, we began to advocate prevention of renal damage by copious oral administration of fluids containing sodium bicarbonate before releasing buried victims. This advice was eventually issued in March 1943 as a Ministry of Health memorandum by Francis Fraser. These measures were often impossible to implement because of local circumstances or more often by the poor instruction and knowledge of rescuers. Some at least survived the initial few hours after release, and once they were in medical hands—in hospital or in first aid posts—intravenous infusion with M/6 sodium lactate with glucose saline, as advised, was sometimes achieved; often it was too late but

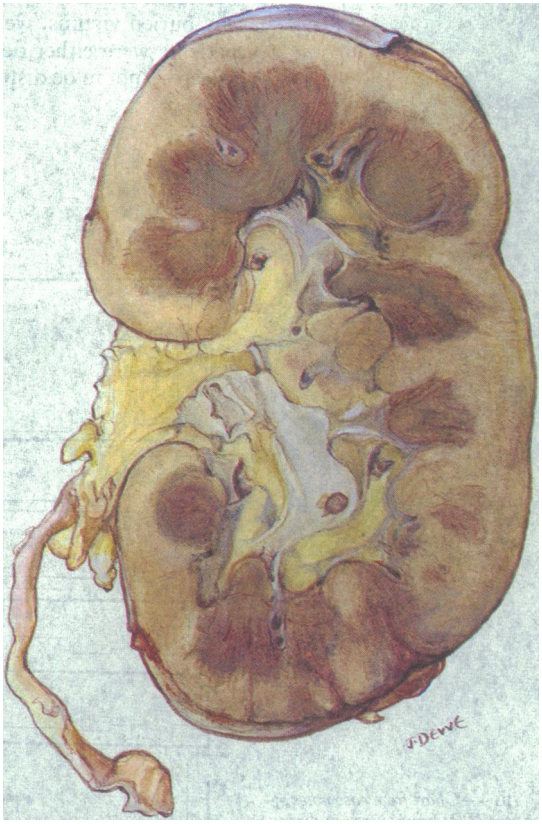

FIG 3—*Kidney of patient who died of uraemia nine days after release; gross swelling (weight 192 g) and pigmented casts*

occasionally it was rewarded by recovery. Such a case was MR (not previously published). The case notes reported:

This woman of 44 (R61 in our recovery series) was pinned down in her house by her legs following a bomb at 12.37 am on 5 July 1944. She was given 1½ pints of fluid before being dug out and admitted (Miss Louden FRCS at the South London Hospital for Women) at 7.10 am. The right leg was blistered with pressure marks, swollen, and weak. 900 ml intravenous M/6 sodium lactate was given and repeated at 4.00 and 5.40 pm. Blood pressure fell from entry level of 142/90 to 100/70 by 5 pm and urine by then was dark red and showed strong bands of oxymyoglobin and the met-compound; pH jugular venous pressure remained low. Oral citrate and bicarbonate with intravenous glucose-saline and lactate were continued over the next three days. Leg circumference measurements were made on both legs at five points and showed maximum swelling between the 7th and 12th day, but fluid intake was good and she maintained good urinary volume. Myoglobin excretion continued until the 6th day and a total of 10 g was measured, equivalent to the total lysis of 2 kg of muscle [fig 4]. Recovery was followed in detail for 13½ months, when blood pressure, urine, blood, and sedimentation rate had returned to normal, leaving some numbness, wasting and loss of power. There was no residual calcification.

This woman's life was saved by prompt and early fluid and alkali administration orally and intravenously. Such an amount of muscle necrosis untreated would have led to irreversible renal failure in those days before dialysis.

A team was formed by the Medical Research Council to try to implement such treatment during the Baedeker raids on provincial towns and cities. Its team leader, Sir James Walton, the eminent surgeon at The London Hospital, and such others that we could muster set off, usually in the dead of night, on priority calls from the Ministry of Home Security to the scenes of disaster—Norwich, Ashford, Weston super Mare, etc, through roads without lighting or signposts, piloted by gallant girls from the services. When we finally arrived at the bombed town with our bottles of citrate, lactate, and glucose-saline, with our syringes and specimen jars and our hand spectroscope, ready to document and treat any buried victims, we found as often as not that the patients were either dead or safely tucked up in bed asleep—"not to be disturbed"—as well as the doctors,

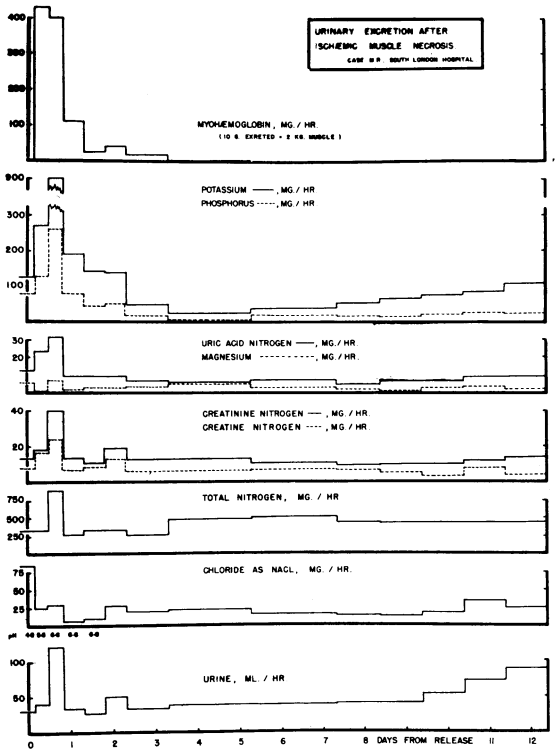

FIG 4—*Chart from case notes of case MR: urine excretion during recovery of 10 g myohaemoglobin (equal to 2 kg of muscle)*

after long and arduous hours of duty. This was not a successful operation. Altogether, however, during the war we recorded a total of 95 recoveries and 98 fatal cases of crush syndrome, largely due to the efforts and cooperation of physicians and surgeons of the emergency medical service. Few were published; there were undoubtedly very many more than we were able to ascertain.

A tragic variant of crush injury happened on the evening of 3 March 1943 when, in an air raid, over 200 people became jammed together for over two hours on the stairs of an underground bombshelter.[14] Many died, and of the 60 who survived and were quickly removed to local hospitals, we recorded 12. One died within 45 minutes of admission from pulmonary and cerebral haemorrhage due to suffocation with widespread ischaemic muscle necrosis but without histological renal change; one showed a blood urea of 380 mg/100 ml (63·46 mmol/l) on day 14 when we saw her but recovered with considerable muscle loss; another of these patients had 1·3 kg of damaged muscle (calculated from her myoglobin output); and others showed lesser degrees of damage and little residual disability.

Other types of anuria associated with myoglobin nephropathy came under renewed scrutiny during this early period of crush consciousness. One woman had femoral artery thrombosis related to cancer and leg muscle necrosis,[15] and there was a young man with "acute post-streptococcal nephritis" in whom autopsy revealed unsuspected muscle necrosis and "crush kidney"; a later elucidated history was compatible with acute and recurrent spontaneous myoglobinuria.[16]

As the result of this work on crush syndrome, and with the temporary cessation of attacks on London, the opportunity arose in 1944 to investigate the extent to which such mechanisms operated in civilian industrial and accidental trauma. The MRC had sent the late Ronald Grant and his "shock research" team from Guy's to Newcastle on Tyne in 1941 to study shock in miners, dockers, and other industrial workers. His team included Basil Reeve, Erasmus Barlow from University College Hospital, and Ludwig Wittgenstein who, resigning his chair of philosophy at Cambridge, had opted for a job as mortuary porter at Guy's. They were concerned with blood loss measurements and replacement, blood pressure, and fat embolism—and Wittgenstein cut beautiful frozen sections of lung. After two or three years they then elected to go abroad to the actual battle scene, at that time Italy; the results of their studies, published later by the MRC,[17] emphasised blood and fluid replacement.

We replaced this team in Newcastle in January 1944. Local good will was already established. In our team were physicians F J Nattrass, C N Armstrong, and F J Ogilvie, surgeons and pathologists, of whom the late Professor Bernard Shaw was the most cooperative and endearing and in whose department our team worked. Perhaps I should particularly mention Miss J K Stead, a hardworking and conscientious chemist whose analyses not only of blood and urine but of dissected individual muscles were essential to our programme. In that brief year by intensive study of 55 cases we established that muscle necrosis played a big part in the shock syndrome that characterised severe industrial accidents. Replacing essential blood does not obviate additional measures to maintain an alkaline urinary flow and to prevent the deposition of acid haematin in the kidney, as is well attested by more recent studies.[18]

One case (notable because it was well followed clinically and biochemically and not hidden under a stultifying diagnosis of "shock" or "traumatic anuria"), was a cholecystectomy patient; both she and the surgeon were considerably obese. The house surgeons, pulling on the retractors for over two hours, produced

necrosis of one rectus abdominis, a comparatively small weight of muscle, and from this the patient died of uraemia a few days later. Compartmental muscle necrosis may have a similar outcome.[19]

The most important finding of this work in Newcastle, however, was the very high incidence of death and of uraemia after resuscitation in patients with fractures of the pelvis. Many muscle fibres (as contrasted with muscle tendons) are attached to the pelvis, and traumatic ruptures of muscle bellies produce considerable ischaemic muscle necrosis due mostly to damage to small blood vessels. Erasmus Barlow and I thought that such cases should be given adequate alkaline fluid on admission, both by mouth and by vein.

By early 1945 we were back in London, then under intensive attack by V1 bombs (doodlebugs) and later by V2s, the silent and stealthy big ones, to put into practice the lessons we had learnt in Newcastle. During these later years we recognised also that renal damage associated with abuse of alcohol or barbiturates was due not to specific kidney toxins but to muscle necrosis from the weight of the unconscious body, as it was in carbon monoxide poisoning. Now 86 causes of myoglobinuria with nephropathy as a contingency are recognised.[5]

In the 50 years since these wartime experiences the incidence of traumatic injuries has increased because of mining, industrial, and automobile traffic accidents, even despite enhanced safety precautions, and earthquakes continue to contribute their regular if sporadic challenge, to which we have offered therapeutic guidelines. The use of simple dialysis by Kolff in his general practice in Kampen during the German occupation of The Netherlands revolutionised the treatment of these potentially reversible renal lesions.[20] The Borst dietary regimen, which spares cell breakdown and potassium release by supplying adequate calories, and our introduction of measures to lower potassium, such as insulin and glucose, have helped. The management of these potentially preventable and reversible cases of anuria after trauma is now well secured by modern dialysis technology (and considerably better than our first struggles with the Kolff machine at Hammersmith in 1946, when only two out of 12 patients survived[21]). The lesson for today is that many of these dialyses for traumatic anuria are avoidable by adequate first aid hydration and alkalinisation as well as by an adequate dietary regimen during recovery.

Finally, tribute should be paid to the Medical Research Council, its scientific workers, and the enthusiastic physicians, surgeons, pathologists, and nurses of the emergency medical service and of the armed forces during the war years—they contributed so much to our knowledge of this subject.

1 Bywaters EGL, Beall D. Crush injuries and renal function. *BMJ* 1941;i: 427-32.
2 Bywaters EGL. Ischaemic muscle necrosis. *JAMA* 1944;**124**:1103-9.
3 Richards NT, Tattersall J, McCann M, Samson A, Mathias P, Johnson A. Dialysis for acute renal failure due to crush injuries after the Armenian earthquake. *BMJ* 1989;**298**:443-5.
4 Collins AJ. Kidney dialysis treatment for victims of the Armenian earthquake. *N Engl J Med* 1989;**320**:1291-2.
5 Knochel JP. Rhabdomyolysis and myoglobinuria. In: Ednoyan S, ed. *The kidney in systemic disease*. 2nd ed. New York: Wiley, 1981:263-84.
6 Bywaters EGL, Dible JH. The renal lesion in traumatic anuria. *Journal of Pathology and Bacteriology* 1942;**54**:111-20.
7 Baker SL, Dodds EC. Obstruction of the renal tubules during the excretion of haemoglobin. *Br J Exp Pathol* 1925;**6**:247.
8 Bywaters EGL, Delory GE, Rimington C, Smiles J. Myohaemoglobin in the urine of air raid casualties with crushing injury. *Biochem J* 1941;**35**:1164-8.
9 Bywaters EGL, Popjak G. Experimental crushing injury. *Surg Gynecol Obstet* 1942;**75**:612-27.
10 Bywaters EGL, Stead JK. The production of renal failure following injection of solutions containing myohaemoglobin. *Q J Exp Physiol* 1944;**33**:53-70.
11 Bywaters EGL. Hydronephrosis: histological resemblances to late changes in the crush syndrome kidney. *Journal of Pathology and Bacteriology* 1945;**57**:394-5.
12 Coupal JF. *History of the medical department of the US army*. Vol 12. Washington, DC: United States Government Printing Office, 1920: 407-577.
13 Bywaters EGL, McMichael J. Crush syndrome. In: Cope Z, ed. *Medical history of the second world war. Surgery*. London: HMSO, 1953:673-88.
14 Bywaters EGL, Crooke AC, Morris CJOR. Limb compression in the tube shelter disaster. *Lancet* 1943;ii:373-93.
15 Bywaters EGL, Stead JK. Thrombosis of the femoral artery with myohaemoglobinuria and low serum potassium concentration. *Clin Sci* 1945;**5**:195-204.
16 Bywaters EGL, Dible JH. Acute paralytic myohaemoglobinuria in man. *Journal of Pathology and Bacteriology* 1943;**55**:7-15.
17 Grant RT, Reeve EB. *Observations on the general effects of injury in man with special reference to wound shock*. London: HMSO, 1951. (MRC special report services No 277.)
18 Better OS, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. *N Engl J Med* 1990;**322**:825-9.
19 Fleischman AH. Ischaemic necrosis of tibialis anticus muscle with renal involvement. *Bulletin of the Hospital for Joint Diseases* 1961;**22**:146.
20 Kolff WJ. First clinical experience with the artificial kidney. *Ann Intern Med* 1965;**62**:608-19.
21 Bywaters EGL, Joekes AM. The artificial kidney: its clinical application in the treatment of traumatic anuria. *Proceedings of the Royal Society of Medicine* 1948;**41**:420-6.

Bell ringers' bruises and broken bones: capers and crises in campanology

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All you that do intend to ring
You undertake a dangerous thing.
(Change ringers' rules, 1694;
All Saints Church, Stamford)

Abstract

***Objective*—To determine the incidence, aetiology, and outcome of injuries due to bell ringing.**

***Design*—Retrospective review of the last six years' issues of *Ringing World*, advertisement in *Ringing World*, and a postal questionnaire sent to 20 active ringing towers.**

***Subjects*—Regular bell ringers.**

***Results*—Seventy nine injuries were identified both from review and by advertisement in *Ringing World*. The incidence of injury among 221 ringers identified by postal questionnaire was 1·8% a year.**

***Conclusion*—Although sonerous, bell ringing can be dangerous and occasionally even fatal. Doctors should be aware of the dangers to which campanologists expose themselves.**

Introduction

Church bell ringing in the British style is an art peculiar to the English speaking world. It is estimated that there are 40 000 bell ringers in England alone and that 3000 towers are rung regularly every Sunday.

Despite the considerable hazards associated with the activity (even the most hardened activists hesitate to call it a sport) little is known about the incidence or range of injuries that occur. As previous medical reports are limited to a single case report[1] and a humorous but instructive article on coping with crises in campanology[2] our aim was to investigate the incidence, aetiology, and outcome of campanological trauma.